

old mucosa and submucosa. This is proven by comparison with the surrounding intact mucosa, which is much less elevated. The microscopical examination shows the thickening to be due to a new development of dense fibrous tissue, such as was described in the later stages of the general pathological process.

There can, I think, be no doubt that the ulcer is of syphilitic origin; the character of the new tissue and the form of cell-death met with seem sufficient proof for this belief. But I think it much more improbable that it is due to the softening of a gumma. Indeed, I find very little evidence in support of such a view. On the other hand, the appearance described speaks more for an indirect form of necrosis of the mucous membrane, brought about by the combined softening of the submucous gummatous infiltration and the obstruction and obliteration of bloodvessels in the same situation. The mucous membrane thus deprived of its nutrition became necrotic, was removed, and the ulcer resulted. The submucosa suffered either directly through necrosis of the infiltrating cells, or, again, indirectly, owing to the vascular changes combined with the action of bacteria.

The clinical course of the disease is made clear by the autopsy findings. The splenic tumor and ascites were the result of the portal obstruction; the obstructing agent was the syphilitic tumor. It seems reasonable to suppose that the gumma was larger at one time than it was at the autopsy, and that the reduction of the ascites was owing to this change. The evidence for this might be found in the size of the left lobe of the liver, and is strengthened by the absence of a marked collateral circulation. The immediate cause of death was the perforation into the peritoneal cavity of the gastric ulcer in a part unprotected by adhesion with surrounding structures.

THE DIAGNOSIS OF NEPHRITIS WITHOUT ALBUMINURIA.

BY ARTHUR R. EDWARDS, A.M., M.D.,

PROFESSOR OF PRINCIPLES AND PRACTICE OF MEDICINE AND CLINICAL MEDICINE, NORTHWESTERN UNIVERSITY MEDICAL SCHOOL; PROFESSOR OF PRACTICE OF MEDICINE AND CLINICAL MEDICINE, NORTHWESTERN UNIVERSITY WOMAN'S MEDICAL SCHOOL; ATTENDING PHYSICIAN TO COOK COUNTY AND ST. LUKE'S HOSPITALS.

THE diagnosis of non-albuminuric nephritis is so inseparably connected, logically and symptomatically, with nephritis attended by albuminuria that certain exceptional features of the latter type must be first considered. Acute and chronic nephritis are usually diagnosed with ease when the urine of every patient is examined systematically, repeatedly, and from twenty-four-hour specimens, considering specific

gravity, gross amount of solid excretion, albumin, and microscopic morphology—casts, blood, pus, etc. It is justly believed that care in the directions indicated insures accurate diagnoses, a supposition in the main correct, and yet subject to various clinical errors. Considering, then, under diagnosis exceptional clinical features, the amount of urine may decrease in the terminal stage of interstitial nephritis, or be constantly small when cardiac hypertrophy fails to develop, for example, in parenchymatous types, or when, in interstitial forms, general nutrition is sufficiently impaired to preclude the usual myocardial hyperplasia and hypertrophy. Low specific gravity and decrease in total solids may indicate persistent functional inadequacy rather than organic renal disease. Albumin is found in most cases in which repeated examination is made of the daily quantity passed. It goes without saying that analysis of single specimens is particularly deplorable from the obvious and often vainly emphasized errors incident to such superficial examination. Clinical experience teaches that we often rely with a sense of false security upon signs and symptoms generally regarded as classical or infallible. Hence we are subsequently astonished at the pathological lesions revealed at autopsy. This statement applies particularly to albuminuria in nephritis. I am certain we overlook both acute and chronic nephritis in regarding albuminuria as a certain or constant symptom. Nephritis without albuminuria certainly exists, although reference to the subject is very meagre in established and current literature.

Casts may be found at intervals when albumin is temporarily absent, when albumin is permanently absent or late in resolving inflammatory processes after chemic tests prove the final absence of albumin. Casts should be searched for, even when albumin is absent, although many are prone to examine the sediment of non-albuminous urine with predetermined negative results.

Our conceptions have been broadened concerning the significance of hyaline casts, now regarded as occurring in urine otherwise apparently normal. Granular and epithelial casts probably always point to degenerative or inflammatory lesions.

As a cardinal diagnostic point with retinitis, albuminuria, and the urinary findings, we consider those cardio-vascular alterations which, frequent in the interstitial, are at least inconstant in the parenchymatous types. Heart and arterial changes are by no means invariable, even in contracted kidney. Cardiac hypertrophy may be simple, as in primary contracted kidney, or eccentric, as in other forms of contracted kidney. There may be dilatation without hypertrophy, or, indeed, even myocardial atrophy is found, as observed in a recent case. The circulatory changes may be otherwise explained — *e. g.*, from arterio-sclerosis of different etiology. Senator states that he who invariably examines the

urine and heart in every instance rarely fails to diagnosticate nephritis. This very interdependence of cardiac and renal physiology and pathology, usually of diagnostic aid, may prove a source of clinical confusion. Thus primary cardiac disease may cause renal congestion, embolism, or even acute or chronic nephritis. Again, alcohol or syphilis may be a common cause for an arterio-sclerosis, myocarditis, and nephritis, diseases as subordinate to the causal factor as possibly independent of each other. Finally, a heart lesion may be wholly secondary to renal disease. While simple renal stasis is usually differentiated with ease by considering the sediment, the absence of inflammatory insignia, and the parallelism between urinary findings and cardiac activity (whence the diagnostic value of such cardiants as digitalis and strychnine), yet in terminal stadia with cardiac weakness, extensive hydrops, dyspnoea, râles, or systolic murmur, it may be difficult or impossible to differentiate between myocarditis with renal stasis and renal disease with ultimate cardiac asystole, although gallop-rhythm is more common in the heart of renal disease than in primary cardiac affections.

The diagnosis of latent, atypical, non-albuminuric nephritis, important in itself, becomes more difficult and important when nephritis is associated with other diseases. In the logical process incident to the diagnosis of frank nephritis, we often ask ourselves, Is the nephritis the sole lesion? Many errors are made in the interpretation of this question, although the observative element in our process may be absolutely correct. Finding a patient with undoubted nephritis we ask, Where does said nephritis stand? What is its exact dignity? Is its cause hidden? Is it primary? Is it somewhat of an accidental finding? At this point the most delicate analysis is demanded if we would diagnose correctly. It is frequently most difficult, or actually impossible, without several early, careful observations of a case, to decide upon the first examination whether a chronic nephritis is the cause of a pericarditis, pleuritis, or pneumonia, since nephritis in lessening physiological resistance is frequently complicated by these highly characteristic, secondary, or it may be, terminal infections. Conversely, a tubercular pleurisy, a pulmonary tuberculosis, or a genuine lobar pneumonia is capable of exciting secondary nephritis. In the two possibilities cited it is not always easy to separate an acute from a chronic nephritis, whence the diagnosis with the parallel prognosis may depend solely upon the chronological test, the clinical evolution of the disease. Many can recall from hospital practice instances of skull fracture, miliary tuberculosis, leptomeningitis, cerebral abscess, sepsis, and other diseases, falsely diagnosed uræmia, simply because nephritis was also present. Senator¹ has said, doubtless from personal diagnostic errors, that with-

¹ Senator. *Erkrank. der Nieren*. Nothnagel's System, Bd. xix. Theil v. Abth. 11, S. 174.

out previous history we can often only diagnosticate a renal disease and suspect other latent affections. The danger of mistaking uræmic symptoms, as hemicrania, nervous symptoms, dysentery, etc., for independent disease must be greater in direct ratio to difficulty of diagnosis. The object of the paper is to emphasize certain anomalous types of both acute and chronic nephritis and personal diagnostic errors in their study. Many authors, as Tyson,¹ have remarked upon the lack of correspondence between the clinical and pathological findings in nephritis. Semmola² holds that clinicians err in considering albumin in the urine the measure of nephritis. Cantharidin produces an intense nephritis, attended, however, by very slight albuminuria, while mercurials produce slight parenchymatous changes with copious albuminuria.

Few authors record acute nephritis sine albuminuria. Lecorché and Talamon have never observed the combination. Quirirolo³ described a case of post-scarlatinal nephritis unattended by albuminuria. In speaking of the general difficulty of diagnosing nephritis when no albumin is found, he remarks that not only œdema, but also uræmia and death occur with wholly negative urinary findings. In his case nephritis was determined at autopsy. *Intra vitam*, the urine was free from albumin; the amount, the specific gravity, and percentage of urea were satisfactory. Dickinson⁴ mentions that casts may be absent in what he designates acute interstitial nephritis, though much albumin be present. Bartels⁵ never observed nephritis without albuminuria in acute parenchymatous nephritis, although in cases of acute scarlatinal nephritis without autopsy he suspected the condition, but could not demonstrate it. Henoch⁶ saw marked acute non-albuminuric parenchymatous nephritis with casts toward the end of life, and in a second case albumin, present at first, later wholly disappeared. Fermi observed a scarlatina epidemic in which nephritis prevailed without albuminuria. We must remember that anasarca complicating scarlatina does not necessarily prove the existence of nephritis. Bartels found casts constantly in acute nephritis. He cautions against interpreting as nephritis the albuminuria occurring in severe infectious disease, even though accompanied by hyaline casts. Blood or blood-casts should also be present to confirm the diagnosis. Rosenstein⁷ possibly hints at the danger in overlooking acute nephritis when he remarks that disappearance of albumin in acute nephritis is no proof

¹ Tyson. *Practice of Medicine*, 1897.

² Semmola. *Nuove contribuzioni sperimentali alla patogenia discrasica o ematogena dell'albuminurica brightica*. *Riforma medica*, 1894, No. 254.

³ Quirirolo. *Riforma med.*, 1896, No. 99.

⁴ Dickinson. *Allbutt's System*, vol. v. p. 369.

⁵ Bartels. *Ziemssen's Pathologie und Therapie*, Bd. ix. p. 247.

⁶ Henoch. *Berliner klin. Wochenschr.*, 1893, No. 50.

⁷ Rosenstein. *Nierenkrankheiten*, pp. 128 and 144.

that the process has ended, since blood and casts may persist and indicate that the inflammation still lives. Rosenstein has seen blood and casts appear in scarlatinal nephritis before albumin could be detected. Senator¹ regards albumin as constantly present in acute parenchymatous nephritis. Leube² has never observed its absence in acute nephritis, although he speaks of alleged instances in literature. Sanné found albumin absent thirty times in one hundred and twenty-four cases of scarlatinal dropsy.

The following case was observed in my service in Cook County Hospital, July 26, 1895, to August 30, 1895:

P. F., No. 143,474. Swede, aged twenty-six years, single. Family history quite negative, one brother having died at twenty-three years with pulmonary tuberculosis. Personal history: Lived in Chicago seven years; drinks beer occasionally; uses no whiskey; smokes. Previous disease includes only poorly-remembered diseases of childhood. Present affliction began three weeks before admission, with anorexia, slight diarrhoea, weakness, fever. No headaches, vomiting, epistaxis.

Examination: Apathetic. Dichrotic pulse. Rose spots. Splenic tumor. Slight diarrhoea, with characteristic typhoid stools. Slight cough. Pulse 70 to 88 or 90. Temperature 102°–104° F., with solution by lysis. Respirations 24. Urine 1175 c.c., acid, amber, specific gravity 1022; no albumin, no sugar, no casts. The diagnosis was typhoid, and the treatment was fluid diet, sponging, salol and calomel. Four days after admission the eyelids became oedematous. The urine showed no albumin. The anasarca becoming very pronounced and notable on chest, ankles, and face, the urine was again examined from a twenty-four hour specimen, with the following result: 1100 c.c.; acid; no reaction to nitric acid, picric acid, or ferrocyanide tests for albumin; specific gravity 1016; microscopically, a small number of red disks, many granular and epithelial casts. The diagnosis of intercurrent acute nephritis in the course of typhoid was inevitable, despite the lack of albumin. Tests were carefully made with the same results again on August 1st, and every other day until his discharge on August 30, 1895, the amount of urine remaining practically the same, specific gravity varying from 1014 to 1016 or 1018. Granular, epithelial, and once several fatty epithelial casts were found. At the time of discharge the casts, so numerous at first, had all but disappeared, an occasional granular cast being found only after examination of several slides. Fortunately, anatomical confirmation of the diagnosis was lacking.

A second case, an acute exacerbation of a chronic nephritis, was seen with Dr. B. Van Hoosen. Previous history was chiefly of repeated attacks of erysipelas covering a period of at least six years. The patient was extremely nervous, and had suffered from weak stomach and capricious appetite. The patient, when first seen, was suffering from recurrent erysipelas, which, nearly healing in one locality, broke out again anew in contiguous areas and covered repeatedly the face,

¹ Nierenkrankheiten in Nothnagel's System specielle Pathologie u. Therapie.

² Leube. Diagnostik der inneren Krankheiten.

trunk, and extremities. Nausea was a prominent symptom. There were no suggestive cardio-vascular findings. The temperature was but little above normal, the pulse tended to rise with exertion. An emphysema, with the usual findings, existed. Nephritis was suspected, but two examinations showed no albumin, no casts, no decrease in total solids, specific gravity, nor quantity. A third specimen gave the least clouding with picric acid; the fourth gave no albumin, but a few hyaline casts, with some blood cells, were detected in the centrifuged sediment. The albumin and casts disappeared, but the general condition became much worse, and in about two weeks after first examination cardiac dilatation with a tachycardia of 130-160 intervened. Cardiac stimulation of enormous amounts of digitalis, morphine, nourishing enemata, infusions of salt solution, whiskey, strychnine, ammonia were unavailing. Just before death considerable blood appeared in the urine. The probable ante-mortem diagnosis was nephritis (acute upon chronic), cardiac dilatation, and recurrent erysipelas. The autopsy, at which Drs. F. X. Walls, B. Van Hoosen, and R. Hickey-Carr were present, revealed very slight arterio-sclerosis, marked pulmonary emphysema, bronchial catarrh and stasis, hepatic congestion, fatty degeneration and focal microscopic induration and round-cell infiltration, induration of spleen and dilated heart. The kidneys were deeply congested, and, while not weighed, measured about 2-4 cm. more than normal in every direction. The capsule was not universally adherent, but tore the renal parenchyma here and there over a considerable area in both kidneys. The cortical markings were obscured, and here, as in the medulla, deep-red streaks alternated with yellow to gray bands. Dr. Walls, Director of the Pathological Laboratory in Northwestern University Medical School, kindly examined microscopically all of the viscera, and found the following changes in the kidneys:

The sections are very diffusely congested. The capsule is thickened and infiltrated with small round cells. Immediately beneath the capsule in the cortex of the kidney are many areas of dense round-cell infiltration which are distinctly perivascular, varying in size from small foci to large wedge-shaped areas about one-fiftieth inch in diameter, with apices directed outward. In the larger areas compressed, degenerated foci are observed, together with atrophied tubules and occasional fibrous glomeruli. The cortex is the seat of advanced parenchymatous change. Little change is observed in the connective tissue, only occasional spots of perivascular round-cell infiltration being seen. Most of the glomeruli are large, with congested tufts and slightly increased connective tissue. Some of the cells of Bowman's capsule are swollen and desquamated. The convoluted tubules are distended, the lumen large, filled with light amorphous, granular material. The epithelial cells are large, coarsely granular, with faint or absent nuclei, often desquamated. Here and there the tubules are filled with red blood-cells. The tubules are relatively intact in the medullary part, although filled now and then with red disks and casts. The large bloodvessels show slight intimal induration and increased fibrous tissue in the muscular coat.

Whether we interpret the pathological findings as those of acute parenchymatous nephritis or as an acute exacerbation of a chronic nephritis, they are equally to the point. In either instance hyaline

casts found once, and a mere trace of albumin, correspond clinically equally well, or indeed better, with parenchymatous degeneration than with acute nephritis of the intensity observed microscopically in the case quoted. I expected to find a chronic interstitial change rather than parenchymatous inflammation.

Rayer¹ was probably the first to remark the disappearance of albuminuria in the course of chronic Bright's disease. Many authors have since noted examples of profound renal lesions without albuminuria during life. Albumin may be absent even in amyloid nephritis. Mahomed states that absence of albumin is almost the rule in the contracted red kidney. Lecorché and Talamon² believe that albumin is rarely absent constantly, that one urinary examination is sufficient, and that tests of several specimens must be made with delicate and various reagents. They quote an instance of saturnine nephritis in which albumin disappeared when the glomeruli became fibrous and their secreting function was suppressed. Burrows described similar cases. Roberts³ relates a case of post-scarlatinal parenchymatous nephritis in which the urine for four months prior to death contained no albumin, blood, or casts. Autopsy revealed a large white kidney. Roberts remarked that the anatomical findings could not be questioned. Lecorché believes that latent nephritis is but nephritis observed in periods of remission or complete compensation. Lecorché and Talamon, after stating the well-known fact that the percentage of albumin is not proportionate to the severity of the renal lesion, describe death occurring from cerebral hemorrhage, cardiac arrest, or intercurrent disease while the urine is quite negative. Necropsy shows contracted kidneys, and the absence of albumin is explained by the fact that the diseased glomeruli become fibrous and impermeable; hence only the sound glomeruli secrete urine, which is therefore free of albumin. Lancereaux⁴ observed fatal saturnine nephritis unattended by albuminuria. Ackermann described a case of chronic parenchymatous nephritis in which, during the last thirteen weeks of life, renal casts were absent. The necropsy explained their absence, since in the pelvis of the kidney there was a mass of casts weighing eight grammes. Eichhorst noticed the absence of albumin from the urine for weeks and months at a time, so that the patient may even be considered well until a fresh setback occurs. Leube cannot confirm statements that albuminuria can be entirely wanting in chronic (interstitial) nephritis, while Roseustein states that albuminuria, though important, is not indispensable, and that we should always examine for casts, since they may occur without albuminuria. Senator maintains that albumin is constant in chronic diffuse non-indura-

¹ Lecorché and Talamon, p. 610. *Traité de l'Albuminurie et du Mal. de Bright.*

² *Ibid.*

³ Roberts on Urinary Diseases, p. 355.

⁴ Lancereaux. *Trans. Internat. Med. Congress*, 1881, vol. xi. p. 191.

tive nephritis, and that cases of alleged interstitial nephritis with wholly non-albuminous urine are but instances of arterio-sclerotic induration. Dieulafoy's experience includes four cases in which the uræmia of interstitial nephritis was unattended for seven months to a year by any albuminuria. Delafield,¹ under the title of "Chronic Productive Nephritis without Exudation," writes that the urine is without albumin or casts or with albumin and very few casts. Bartels states that the chemic analysis shows constant albuminuria in chronic parenchymatous nephritis, which never fails during the entire clinical course. The same author holds that it is not constant in interstitial types, and observed a single instance in which albuminuria was constantly absent. Tünger has often found it absent in chronic interstitial nephritis. Regarding absence of casts, Sehrwald² noted disappearance of casts without that of albumin. He described in urine containing but few casts many granular masses and free nuclei, while the cylinders became paler and more delicate. Casts, he maintains, may be dissolved in urine, not from chemic causes nor from decomposition alone, but from the presence of pepsin in acid urine, the action of the pepsin being increased by the urine remaining long in the bladder or by high temperature. The dissolution of casts may occur even in the kidney itself. He recommends examination of freshly-catheterized specimens, not long excreted into the bladder before withdrawal.

CASE III. *Acute diffuse parenchymatous nephritis*.—W. A. W., St. Luke's Hospital, October 25 to November 4, 1896. History written by Dr. Thomas H. Lewis. A book-keeper, English nativity, aged thirty-five years, married. Complains of pain in back, chest, and legs. On September 26, 1896, present illness began with dull pain extending from throat to epigastrium, and with pain in the lumbar region. Marked dyspnœa and palpitation existed. There was pain over the lower lobe of the left lung. Great thirst, anorexia, constipation. After treatment outside of hospital, pain disappeared and dyspnœa ceased; but two weeks later, after sleeping near an open window, his joints became swollen, the left knee being especially painful.

Previous illness: Usual diseases of childhood. At seven, rheumatism. In 1890 stomach trouble from which patient lost seventy-five pounds. The thyroid gland became swollen, his pulse rapid, and marked exophthalmos developed. No history of venereal disease.

Family history. Maternal grandfather died of gout. Otherwise negative.

Personal history. At present uses tobacco and liquor moderately, although formerly drank whiskey in large amounts.

Physical examination. General nutrition fair. Mucous membranes normal. Considerable exophthalmos, with von Graefe's sign. Tongue slightly coated. Lungs negative. Dilatation of left ventricle, apex being finger's-breadth beyond the nipple-line. A short, superficial,

¹ Delafield. Twentieth Century Practice, vol. 1.

² Sehrwald. Deut. med. Wochenschr., 1890, No. 24.

rather rough systolic murmur was heard during complete expiration during the last two days of life. The pulse was uniformly rapid, from 110 to 120. Respirations 38 to 44. Temperature-curve ranging from 101.6° to 103.8° F., fairly though not absolutely continuous. The thyroid was not enlarged. Abdomen negative, no tympany, rather sunken in contrast with the well-formed thorax. No splenic tumor; liver, normal outlines. The knee-joints were swollen, painful, with distinct floating of the patella. Pain without swelling in right shoulder and left wrist. No œdema. The patient complained of precordial pain. Alkalies and salicylates, and, finally, potassium iodide in full doses relieved neither the pain nor the swelling. The bowels moved freely after administration of calomel; stools were dark in color. Considerable pharyngeal pain and hyperæmia on the ninth day; appropriately treated. At the same time the pulse rose to 150, and dyspnœa, with cyanosis, was marked; combated with strychnine, digitalis, camphor, and whiskey. Death from cardiac exhaustion on the eleventh day after entrance. Urinalysis, acid, 1018 to 1028, darkish color, no albumin, no sugar, urea 2 per cent., amount thirty to forty ounces, although bowels were constantly relaxed; microscopic examination showed stray pus-cells.

The diagnosis was uncertain for a long time. The patient had exophthalmic goitre, which might have explained many of the findings, yet certainly not the temperature, angina, and articular signs. Typhoid fever was excluded by the absence of roseolæ, splenic tumor, tympany, character of the stools, absence of delirium, etc. A rapid pulse in a goitrous subject would not exclude complicating typhoid. Miliary tuberculosis was considered, favored by the cyanosis, dyspnœa, and increased respirations, perhaps, but still the circulatory failure, articular swellings, angina, argued against it. Sepsis cryptogenetica best explained the course and findings, the angina, synovitis, etc. Ulcerative endocarditis was considered, but rejected. The urine was searched chemically and microscopically with great care for possible evidence corroborative of sepsis in the form of nephritis, but repeated examinations were negative.

Autopsy, November 6, 1896. Pleuræ practically negative, except many ecchymoses. Pericardium-cavity obliterated almost completely by old adhesions and by fresh exudate at apex, mostly fibrinous, but partly sero-sanguineous. Stripping off the fresh exudate, the visceral and parietal leaves of the pericardium showed hemorrhagic areas. The valves were practically normal. The ventricles were dilated and hypertrophied, the right being 6 mm. and the left 15 mm. thick. The lungs showed a few hemorrhagic infarcts, pulmonary œdema, hypostatic congestion, and a few cases of lobular pneumonia. Hemorrhages were found in the renal pelvic mucosa, in the pancreas, and in the articular synovial membranes. The liver was slightly enlarged, fattily degenerated, and somewhat congested. The kidneys were very large; unfortunately, they could not be weighed; the capsule stripped readily, showing subcapsular ecchymoses. The cortex was swollen and its markings were very indistinct. The organs were the seat of cloudy swelling, and were, like other parts, considerably congested. Several white infarcts existed. As the macroscopic renal findings were a surprise, special attention was given to the microscopic examination. In the cortex were areas in which exudation of white cells was sufficient

to obscure the field; less exudation existed in the medulla. The glomeruli were not greatly altered, there being some congestion, some exudation, and considerable deformity by thickening of the capsule. The epithelial cells lining the large and small tubules were universally degenerated, disintegrated, and desquamated, the cells without nuclei contrasting sharply with the deeply-stained extravasated leucocytes. No essential nor systematic increase in connective tissue, except in quite a number of the glomeruli. Undue vascularity existed, together with pouring out of the hæmocytes into the desquamated uriniferous tubules.

Diagnosis. An acute diffuse parenchymatous nephritis, with possibly more ancient changes of distinctly minor dignity.

Stewart¹ justly remarks that when albumin is not found, the later search for casts is at the best perfunctory. Said search must be conducted carefully with the aid of a centrifuge, with a low lens and properly graduated illumination. He believes that albumin may be constantly absent throughout the course of chronic nephritis, and casts be detected only after much search. He frankly states the conviction, which we also feel regarding our own experience, that he has overlooked many cases of nephritis without albuminuria. Wilkes² has pointed out that granular or cirrhotic kidney may not be evidenced by albuminuria, Stewart admitting that the form described is not a true nephritis. As Stewart states, Osler, Fagge, Jaksch, and others mention the subject briefly, if at all. Millard³ and Mahomed⁴ speak of the absence of albuminuria, but refer chiefly to granular kidney or the typical arterio-capillary fibroid kidney of Gull. In the cases reported by Mahomed, the kidneys belonged to the second stage of his classification, the first being functional renal inadequacy, without renal or circulatory alteration, the second chronic Bright's disease without nephritis (red granular kidney, arterio-capillary fibroid kidney), and the third chronic Bright's disease with nephritis (mixed or mottled granular kidney), where epithelial changes exist and diagnosis is easy. Stewart's estimation of Mahomed's view as extreme is just. It seems that Mahomed has, in instances at least, confused cause and effect, and that arterio-sclerosis is forced to explain too much.

Case I. of Stewart was nephritis without albuminuria, but with hyaline and granular casts found once. Of his seven cases one came to autopsy, which revealed granular kidneys and hypertrophied heart, although the casts and albumin seemed to have been absent throughout its course. Stewart holds that several of his cases do not correspond

¹ Stewart, D. D. Occurrence of a Form of Chronic Bright's Disease, other than Typical Fibroid Kidney, without Albuminuria. Transactions Pan-American Med. Congress, 1893, p. 191. See ref. 23-24.

² Wilkes. Guy's Hospital Reports, second series, vol. ii.

³ Millard. Chronic Bright's Disease.

⁴ Mahomed. Guy's Hospital Reports, 1879, vol. xxiv.; and Chronic Bright's Disease without Albuminuria. Ibid., 1880-81, vol. xxv.

to the fibroid kidney, the one form in which albumin may be persistently free. Epithelial changes were present, proven from the finding of hyaline, granular, and waxy casts.

He tabulates the differences between what we may term his type of nephritis and the parenchymatous and interstitial forms as follows :

<i>Chronic Parenchymatous Nephritis.</i>	<i>Chronic Interstitial Nephritis.</i>	<i>Form of Chronic Non-albuminuric Nephritis closely resembling neither.</i>
Urine always albuminous; scanty in amount except during secondary atrophy, then may be abundant; light colored, depositing urates readily. Specific gravity normal or lighter than normal, though low for amount passed.	Urine not constantly albuminous; usually to degree recognizable by Heller's test. Urine profuse unless cardiac failure with dropsy, etc., light colored, low gravity; slight sediment, often invisible.	If urine is ever albuminous, it is probably not as an incident of the malady, as the most decided symptoms of renal inadequacy may be present, or indeed uræmia with persistent absence of albumin; urine subnormal in amount, quite scanty, high colored, without cardiac weakness and dropsy, with high blood-pressure; gravity lower or higher than normal, though relatively low considering amount.
Casts numerous, great variety, epithelial, granular, hyaline, waxy; blood-corpuses, and connective tissue; shreds not infrequent. Microscopically urates and phosphates predominate; oxalates less common.	Casts not frequent, usually hyaline, less often granular. Renal epithelium and blood-cells scanty or absent; oxalates common.	Casts common, but not numerous, principally hyaline but granular, present in all though in less number; epithelial casts rare (seen in very small number in two cases); waxy casts in one case; cylindroids in all, in several very numerous; renal epithelium in all; oxalates very common; urates often in relative excess; occasionally blood and pus cells.
Urea usually much diminished, uric acid practically normal.	Total solids often normal; urea may be normal, usually diminished; uric acid is usually diminished.	Total solids, urea and mineral salts, always diminished in amount; uric acid normal or diminished.
Cardiac hypertrophy not invariable, though tendency to increased blood-pressure. Atheroma not common in early stages.	Hypertrophy usual; blood-pressure raised; fibrosis almost invariable and pronounced.	Hypertrophy not detected, although in all first sound forcible, second aortic accentuated; blood-pressure raised in five (out of seven); not detectable in two while on non-nitrogenous diet; no arterio-sclerosis in any.
Uræmic symptoms common, although less than in interstitial; no loin pain.	Uræmia common; no loin pain.	Uræmic signs common; loin pain usual.
Dropsy usual; obstinate.	No dropsy, and even œdema not often detectable till disease advanced.	No dropsy; œdema not common.
Occurs mostly after age of forty. Patient pale and waxy looking.	Mostly after forty (degenerative period); for a long time only failure of nutrition noted; grayish complexion.	Three cases under thirty, others middle aged; no degenerative process; facies pale; no marked anæmia.

Stewart comments upon the dissociation of casts and albumin in his series of cases, casts being more or less constantly present, while albu-

min was usually absent. Probably casts are due "rather to faulty metabolism or inflammatory irritation of the renal epithelium than to exudation of serum."¹

Charles W. Purdy,² of Chicago, wrote an exhaustive article upon the "Pre-albuminuric Stage of Chronic Nephritis," in which, however, his stress was laid particularly, as his title hints, upon the absence of albuminuria in the formative stages of the disease, quoting Burrows, Christison, Rayer, Reed, Malmsten, Grainger Stewart, and Roberts.

Graves and Monneret have noted nephritis without albuminuria, as have also Hue, Huchard, Millard, Guesdon, Dolespierre, Gand Delalande, Dieulafoy, de Coquet, and others already cited.³

We are forced to ask ourselves whether casts always indicate nephritis—*i. e.*, whether finding casts without albumin is adequate proof of nephritis. Zimmermann, Key,⁴ Griesinger, Meyer,⁵ Rosenstein,⁶ Vogel, Thomas,⁷ Senator,⁸ Burkart,⁹ Fischl,¹⁰ Bartels,¹¹ Nothnagel,¹² etc., have exceptionally observed cylindruria without albuminuria. When Steinbeck constructed the centrifuge, Leyden and Glaser found hyaline casts in the urine of healthy individuals. Radomyski¹³ examined cases of valvular heart disease, arterio-sclerosis, gastro-intestinal inflammation, pulmonary tuberculosis, acute infectious diseases, carcinoma, cachexia, nervous diseases, and healthy individuals, and found casts in a very great number, explained by him as the result of circulatory disturbance. Daiber¹⁴ found hyaline and granular casts without albumin, especially in circulatory and amyloid disease. Alber¹⁵ reported granular, hyaline, and epithelial casts and red blood-disks without albuminuria. Kossler¹⁶ reports hyaline, granular, epithelial, waxy, and corpuscular casts in many infectious and other conditions without coincident nephritis or albuminuria. The specimens microscopically showed only degenerative changes, but no inflammatory insignia. In his observations nucleo-

¹ Lubarsch. *Centralbl. f. allgem. Path.*, Bd. iv. No. 6; Aufrecht, *Centralbl. f. clin. Med.*, June 30, 1893.

² Purdy. *Pre-albuminuria Stage of Chronic Bright's Disease*. Chicago Medical Journal and Examiner, May, 1885.

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⁷ Thomas. *Archiv f. Heilkunde*, Bd. xiv.

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¹¹ Bartels. *Ziemssen's Pathologie und Therapie*, Band ix. p. 247.

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albuminuria and cylindruria occurred together, whence he distinguishes two types of cylindruria, (*a*) degenerative and (*b*) inflammatory. He has observed in scarlatina, as had Baginsky in diphtheria, febrile albuminuria, followed by cylindruria with nucleo-albuminuria and, finally, by nephritis with albuminuria. In the past four years I have observed three cases of generalized fleeting anasarca in which direct examination, as well as argument by exclusion, led me to diagnosticate angio-neurotic oedema. In one instance, seen with Dr. T. J. Watkins, a healthy man suddenly complained of swelling of the eyelids, and the next morning of swelling of the penis and scrotum. There was no venereal history nor lesion. A few hours later the feet, wrists, body, and lips swelled to an enormous extent. Examination of the urine gave acid reaction, 1020, normal quantity, and solids, no albumin, etc., and no casts nor formed elements in the fresh and centrifuged sediment. A circum-spect diagnosis of angio-neurotic oedema was made. Recovery was complete in a few days. The case is presented without comment other than that it might have been a nephritis without cylindruria and albuminuria.

The chief considerations and conclusions of the paper may be summarized as follows:

1. Carefully repeated routine chemical and microscopical examination of the urine every twenty-four hours usually, but not invariably, detects acute and chronic nephritis.
2. The diagnosis of the albuminuric and non-albuminuric types of the nephritides is aided by searching examination of other viscera and parts, *e. g.*, by disclosure of cardio-vascular changes, retinal involvement, etc.
3. These visceral or somatic changes, usually present in nephritis, may be lacking in concrete instances or be capable of other or diverse interpretation, as polyuria, atheroma, etc.
4. The urinary findings, most essential to the diagnosis of nephritis, may be lacking, as may other signs and symptoms of minor dignity. Hence, as we fear instinctively, as it were, the existence of nephritis in certain cases before we examine the urine, so we may still fear its existence after negative urinalysis.
5. Nephritis may be unattended by albuminuria. Such nephritis is usually interstitial in type, of which cases abound in literature.
6. While certain instances of non-albuminuric nephritis correspond to the type described by Dr. D. D. Stewart, yet non-albuminuric nephritis may not exactly correspond to said type, since acute nephritis, chronic parenchymatous nephritis, and chronic interstitial nephritis may exceptionally occur without albuminuria.
7. Casts should always be searched for; they are more constantly found than is albumin, yet they seem in certain instances to betoken

renal degeneration rather than inflammation. They are not invariable in nephritis nor are they invariably nephritic.

8. Future clinical caution and pathological examinations will probably increase the number of cases of non-albuminuric renal inflammations of acute, subacute, and chronic types.

9. Non-albuminuric nephritis is of especial importance (*e. g.*, Stewart's type) in life-insurance and kindred examinations and in practice, since prophylactic measures may be instituted and the prognosis obviously influenced.

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THE SO-CALLED HYALINE BODIES AND OTHER CELLULAR DEGENERATIONS IN NASAL POLYPI.

BY JONATHAN WRIGHT, M.D.,
 OF BROOKLYN.

In a paper (*New York Medical Journal*, November 13, 1897) read before the American Laryngological Association in 1897 I had occasion to refer to a case of adenoma of the nose occurring in the practice of Dr. F. W. Hinkel, of Buffalo. He had sent me a slide holding several microscopic sections from this rare nasal growth. These sections had been stained only with hæmatoxylin, and, although they served the purpose of illustrating certain phenomena with which that paper was concerned, they did not present the striking pictures of those objects with which this communication deals. The growth recurred, and Dr. Hinkel has sent me the portions removed by him, and for thus giving me an opportunity again for a study which is, at least to me, very interesting, I am deeply indebted to him.